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Thromboangiitis Obliterans in a Woman: Factor of a Mild Electric Shock in Initiating Gangrene

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ALTHOUGH the reason cannot be satisfactorily explained, thromboangiitis obliterans (Buerger's disease) in women[1] is rare. Other peculiarities of this disease are its rarity in Negroes,[2, 4] its rare association with diabetes mellitus[6] and its relative preponderance in individuals of Jewish ancestry.[3, 8]

Silbert[5] in reviewing his own series of 1,400 cases of thromboangiitis obliterans found only 12 cases clinically diagnosed in women. The Mayo clinic group[1] in a survey of the literature in 1946 found only 20 reported cases in women, with only four of the 20 proven by microscopic section. Eight were typical cases and eight were apparently not thromboangiitis obliterans. Davis and King[4] reported the only proven case in a Negro woman.

In the diagnosis of thromboangiitis obliterans, care must be taken to exclude the instances of peripheral vascular disease due to phlebitis, embolism, polycythemia and ergot poisoning, and of arteriosclerosis or arteritis due to other causes such as syphilis, periarteritis nodosa, disseminated lupus erythematosus and disseminated arteritis. In arriving at a diagnosis of thromboangiitis in a woman, it should be established that she is a smoker of tobacco, and the following features should be present:

1. Evidence of organic occlusion of the large arteries of the extremities.
2. Onset of symptoms relatively early in adult life.
3. Involvement of arteries in both upper and lower extremities.
4. Absence of demonstrable arteriosclerosis, diabetes mellitus and other causes of peripheral vascular disease.

The following report of a case is submitted not only because of its occurrence in a woman but because of the unusual exciting agent which apparently precipitated the gangrenous process.

REPORT OF A CASE

A white married woman, aged 34, of Dutch-English ancestry, ten years previously had first experienced intermittent aching pains in the fingers of the hand upon exposure to cold, associated with color changes of pallor, blueness and redness. These symptoms gradually increased in severity. Early in 1945 she visited a clinic in the East where Raynaud's disease was diagnosed. Eighteen months before the present illness, pains upon exposure to cold appeared in the toes of the feet, and mild intermittent claudication was felt in both legs upon walking two to three blocks at a normal gait. The patient said she had smoked a minimum of one package of cigarettes daily for many years. There was no history of injury to an extremity or of arthritis, diabetes mellitus, lues, or other contributory disease, and the patient said she had never had thrombophlebitis. There was nothing indicative in the family history.

Present illness: The patient, a telephone switchboard operator, while pulling cords from the switchboard felt pain in the distal portions of the second and third fingers of the left hand, as if strands of wire had pierced the skin. After a few days she reported for medical care. The examining physician's findings one week after the injury were a puncture wound of the fat pad of the left index finger and cellulitis. A wet dressing was applied and the finger was immobilized with a splint. The puncture wound continued to drain, and penicillin was administered daily. The condition remained subacute for the next two months, although the adjacent middle finger became involved in the inflammatory process. Conservative treatment was continued until approximately ten weeks after the initial complaint, when the patient was hospitalized, and an amputation was performed through the proximal phalanges of the index and middle fingers. Healing was satisfactory. The pathologist's report of the microscopic sections (Figures 1 and 2) was: Obliterating endarteritis of the small blood vessels. Some of these were completely obliterated. There was some perivascular infiltration of the ground cells. Diagnosis: Thromboangiitis obliterans.

The results of general physical examination, including all indicated laboratory tests, were essentially negative. There was no evidence of scleroderma, scalenus anticus syndrome or cervical rib. Peripheral arterial pulsations recorded a week before amputation were as follows: The radials and posterior tibial artery pulsations were palpable,

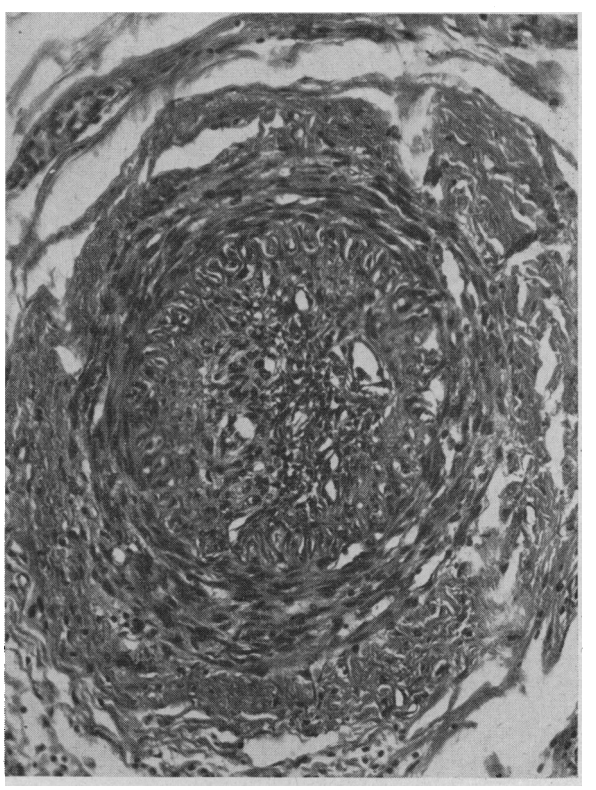

Figure 1.—Digital artery (x 200). This closely resembles Figure 95, "Old healed stage in small artery," in Buerger's text, "The Circulatory Disturbances of the Extremities," p. 343.

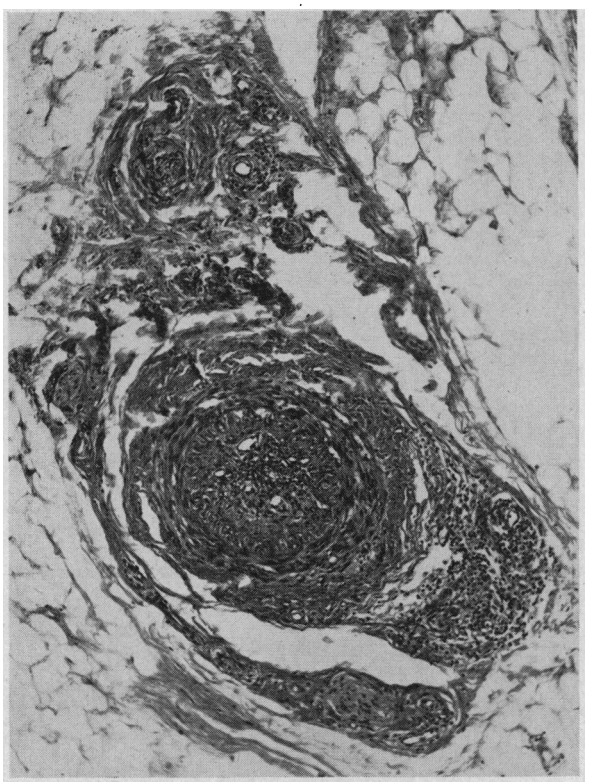

Figure 2.—Low power view (x 90) from section where Figure 1 was obtained.

but pulsations in the ulnar and dorsalis pedis arteries could not be felt. The patient, who had continued to smoke, was reexamined ten months later. The amputation sites were healed. The fingers and toes remained painful on exposure to cold, with color changes in the fingers, and there was still complaint of mild intermittent claudication in both calves on walking two to three blocks at a normal pace. The fingers were cold and dry, as were the feet. There were no postural changes. Palpable peripheral pulsations were as follows: The right dorsalis pedis was faintly palpable, but the left dorsalis pedis and both posterior tibials could not be felt. The popliteals and radials were normal and the ulnar arteries were not palpable.

COMMENT

The early diagnosis of Raynaud's disease in this case is understandable. The early symptoms were of a vasospastic character and resembled those of Raynaud's syndrome. Thromboangiitis obliterans was apparently not considered due to the lack of signs and symptoms of organic arterial occlusion. The subsequent course of events led to the correct diagnosis. Practically all the diagnostic criteria were present in this case, even the factor of smoking. The trauma which initiated the gangrenous process was unusual and open to controversy. As there were no strands in the telephone cable in question, the possibility of perforation of the skin by fine strands of wire was eliminated. It was found that 0.70 to 0.71 amperes at 24 volts direct current was periodically present about the telephone plugs and a mild shocking current could be felt, especially if the subject had moist hands. It is felt that a sufficient electrical current was present to cause cellular damage. Although the trauma that such a current might cause to normal tissues would be practically unnoticed, ischemic tissues, such as those present in this case, frequently respond poorly even to trivial trauma. This may have been a significant factor in this case.

CONCLUSIONS

A case of proven thromboangiitis obliterans in a white woman is reported.

An electric charge of 0.70 to 0.71 amperes at 24 volts direct current may have been the traumatizing agent in this case. Apparently sufficient damage was done to the ischemic tissues to precipitate a gangrenous process.

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The Use of BAL in Generalized Argyria

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HEAVY metal poisonings, especially the toxic effects resulting from gold,[2, 10] arsenic,[2] and mercury,[1, 4] have been successfully treated with BAL (British Anti-Lewisite, 2, 3-dimercaptopropanol). This drug was also discovered to be efficacious as an antidote in cadmium,[9] zinc,[13] copper,[6, 7] lead,[12] antimony, bismuth, chromium, and nickel poisonings.[1] No previous publication has been noted concerning the use of BAL in clinical argyria. The successful management of silver poisoning, whether local or generalized, has always remained a therapeutic problem. With the efficacy of BAL in other heavy metal toxicities a trial with this drug appeared indicated in a case of long-standing generalized argyria.

Arsenic, gold, and mercury, in particular, produce their toxic effects by combining with the sulfhydryl groups of tissue proteins of cellular enzymes to form mercaptides, thereby disrupting certain vital physiological processes. The sulfhydryl radical in dithiol BAL competes with the dithiol protein-metal compounds, thereby separating the offending metal from tissue union. To be effective, BAL must be administered soon after a heavy metal combines with the sulfhydryl group, otherwise the effect of the metal becomes irreversible.[10]

The distribution and metallic retention of silver in the body is very different from that of gold, arsenic, and mercury in the tissues. There is specific affinity of silver granules for the connective tissue framework and vascular system. In cases of argyria the reticulo-endothelial system is the site of the initial deposition, following which the majority of body structures contain silver deposits.[5] Silver, which is deposited as a colorless substance, is uniformly distributed in the corium and darkens as the result of light influence.[11] This metal so deposited remains chemically unchanged or is oxidized as silver oxide or silver sulfide, depending on the loca-

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